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Train Stimulation at the Atria for Prevention of Atrioventricular Tachycardia: Dependence on Accessory Pathway Location

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In 12 patients with accessory pathway-mediated supraventricular tachycardia, programmed electrical stimulation with a rapid train of 10 stimuli was assessed for prevention of tachycardia induction. Tachycardia was induced with one or two extrastimuli from both the right and the left atrium (by way of the coronary sinus). Preventive train stimulation, with the train delivered after the tachycardia-initiating stimulus, was attempted at the site of tachycardia induction as well as at the opposite site. Prevention at the site of tachycardia induction was successful in all patients when the length of the train (90 ms) exceeded the effective refractory period of the tachycardia-initiating stimulus to achieve single atrial capture within the "preventive zone." However, in patients with a left-sided accessory pathway, preventive stimulation at the right atrium failed when tachycardia was induced from the coronary sinus because of interatrial conduction delay.

It is concluded that train stimulation is an effective mode for supraventricular tachycardia prevention, yet the site of preventive stimulation should lie as close as possible to the anatomic site of the reentrant circuit to reduce interatrial conduction delay.

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The induction as well as maintenance of a tachycardia based on the reentry phenomenon depends on a delicate balance between conduction velocities and refractory periods within the two limbs of the reentrant circuit. Electrical interference with these properties by premature stimulation will terminate an ongoing tachycardia and is the operating principle of antitachycardia pacemaker devices (1). However, well timed premature stimulation may also interfere with, and ultimately prevent, the induction of a reentrant tachycardia, as has previously been shown (2). To suppress the onset of atrioventricular (AV) tachycardia, an atrial extrastimulus must be delivered after the tachycardia-initiating stimulus within a critical time interval immediately after the atrial effective refractory period of the initiating stimulus (Fig. 1A and B). This interval is termed the "preventive zone." Any stimulus delivered within the preventive zone will interfere with the buildup of the reentrant circuit initiated by the preceding stimulus and thus prevent tachycardia induction. Stimuli delivered after the preventive zone reset the tachycardia.

We assumed that, to circumvent the need to critically time the preventive stimulus, several stimuli delivered in rapid succession should also prevent tachycardia, provided they are so delivered that only one of them achieves atrial capture within the preventive zone. Our study addressed several questions to further assess the validity of the preventive mode of stimulation: 1) Will stimulation for prevention of AV reentrant tachycardia remain effective if, instead of a single extrastimulus, a rapid train of extrastimuli is delivered? 2) To what extent does the anatomic site of tachycardia induction influence preventive stimulation? 3) Can right atrial stimulation prevent tachycardia mediated by a right-sided as well as by a left-sided accessory pathway?

Methods

Study patients. Twelve patients with symptomatic AV reentrant tachycardia undergoing standard electrophysiologic investigation for evaluation of tachycardia characteristics and mode of therapy were studied (Table 1). There were six men and six women with a mean age of 34 years (range 16 to 65). Six patients had a right-sided and six a left-sided accessory pathway; in three patients the accessory pathway revealed concealed conduction only. Before the electrophysiologic investigation all patients had been without medication for at least 72 hours. Informed written consent was given by all patients.

Electrophysiologic study. Using the Seldinger technique, quadripolar catheters were inserted through the fem-

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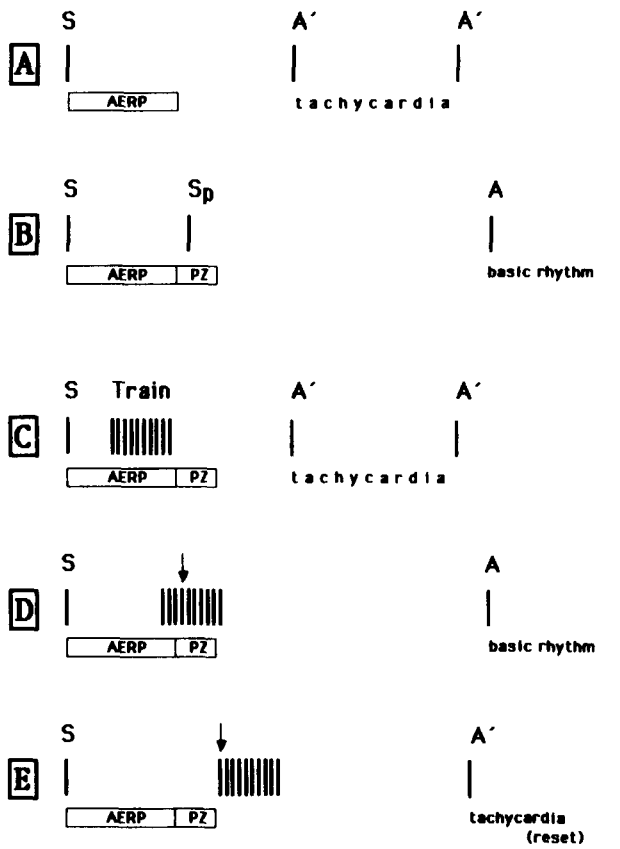


Figure 1. Schematic showing principle of preventive stimulation. **A,** An extrastimulus (S) induces tachycardia (initial tachycardia beat is termed A'). **B,** A single stimulus (Sp) delivered within the preventive zone (PZ) prevents tachycardia induction: basic rhythm is resumed with a beat termed A. Note that the preventive zone follows right after the atrial effective refractory period (AERP). **C,** A train of stimuli remains ineffective when delivered before the preventive zone within the atrial effective refractory period; tachycardia is induced. **D,** Tachycardia induction is prevented when the stimulus train extends beyond the atrial effective refractory period into the preventive zone. **Arrow** indicates single train stimulus to achieve atrial capture. **E,** Tachycardia is reset when the train is delivered outside the preventive zone. **Arrow** indicates the single train stimulus to achieve atrial capture.

oral veins in the high right atrium and in the coronary sinus; bipolar catheters were placed across the tricuspid valve to record His bundle electrograms and at the right ventricular apex.

Stimulation protocol. The assessment of basic electrophysiologic variables in patients with an accessory AV pathway, as performed in our laboratory, has been described in detail (3). Effective refractory periods of the accessory pathway in the anterograde direction and in the right and the left atrium (by way of the coronary sinus) were determined in the setting of the induction mode of AV reentrant tachycardia.

Tachycardia was induced by decremental pacing with a maximum of two extrastimuli during sinus rhythm or during basic drive pacing of the right atrium at 640 and 510 ms. Tachycardia induction was attempted at the high right atrium as well as the coronary sinus. Programmed stimulation for termination of tachycardia included a maximum of three right atrial extrastimuli or, if that approach failed, a maximum of two right ventricular extrastimuli.

When the efficacy and reproducibility of the tachycardia induction and termination modes were ascertained, preventive stimulation with a train of 10 extrastimuli, 10 ms apart, was begun. With tachycardia initiated at the high right atrium, preventive stimulation was performed at the same site; with tachycardia initiated at the coronary sinus, preventive stimulation was performed at both the high right atrium and the coronary sinus. The train of stimuli was initially delivered 50 ms after the tachycardia-initiating stimulus and was then progressively delayed in 10 ms steps toward diastole. We expected that, as long as the duration of the train (90 ms) was encompassed within the atrial effective refractory period of the tachycardia-initiating stimulus (Fig. 1C), tachycardia induction would not be affected. However, as soon as the train was delayed to extend beyond the atrial effective refractory period (Fig. 1D), tachycardia would be prevented by atrial capture of the first train stimulus to fall into the preventive zone. Tachycardia would be reset by atrial capture if the train was delivered outside the preventive zone (Fig. 1E).

Equipment. Single extrastimuli at twice diastolic threshold and 0.5 ms duration were provided by an ERA-S-HIS stimulator (Biotronik GmbH, Berlin); trains of stimuli at twice diastolic threshold and 0.5 ms duration were provided by the Medtronic SP0503MKIV stimulator. Three surface electrocardiographic (ECG) leads (filtered at 0.1 to 20 Hz) and five intracardiac leads (filtered at 40 to 500 Hz) were simultaneously recorded on magnetic tape and paper (100 mm/s).

Results

Tachycardia induction. Relevant electrophysiologic data are given in Table 1. Atrioventricular tachycardia of the orthodromic type was inducible in all patients from the high right atrium (during sinus rhythm in Cases 4 and 8 to 12; at a basic cycle length of 640 ms in Cases 1, 3, 5 and 7; and at a basic cycle length of 510 ms in Cases 2 and 6). In 10 patients tachycardia was also inducible from the coronary sinus (during sinus rhythm in Cases 7 to 12; at 640 ms in Cases 1 and 5; and at 510 ms in Cases 4 and 6). With one exception, a single extrastimulus was required to induce tachycardia from the right atrium, whereas two extrastimuli were necessary to induce tachycardia from the coronary sinus in five patients. The mean tachycardia cycle length was 328 ± 41 ms. Tachycardia could be terminated in five patients (Cases 2, 3, 7, 8 and 12) from the high right atrium and in the remaining seven from the right ventricle.

Table 1. Electrophysiologic Data in 12 Patients

Patient	Age (yr) & Sex	Diagnosis	Site of AP	Effective Refractory Period (ms)			Conduction Time (ms)		Initial Tachycardia Interval at Site of Induction (ms)		TCL (ms)
				AP (ant)	HRA	LA	HRA → LA	LA → HRA	HRA	CS	
1	24M	WPW	Left post lat	260	230	230	100	100	430	380	300
2	17M	WPW	Right lat	260	160	210	130	140	480	—	340
3	65M	WPW	Right sept	290	160	310	120	120	630	—	410
4	16F	WPW	Left lat	290	240	270	90	90	480	370	360
5	53F	CAP	Right lat	—	240	220	110	110	370	400	330
6	44F	WPW	Left post lat	270	210	220	90	90	410	330	320
7	26F	CAP	Left post sept	—	190	270	70	60	480	270	290
8	22M	CAP	Right sept	—	250	330	110	120	400	420	350
9	48M	WPW	Left lat	220	200	260	130	130	500	360	320
10	25M	WPW	Right sept	280	240	280	120	100	550	470	360
11	28F	WPW	Right sept	290	230	280	100	110	340	390	250
12	45F	WPW	Left post lat	280	210	270	110	90	530	290	310

ant = anterograde; AP = accessory pathway; CAP = concealed accessory pathway; CS = coronary sinus; F = female; HRA = high right atrium; LA = left atrium; lat = lateral; M = male; post = posterior; sept = septal; TCL = tachycardia cycle length; WPW = Wolff-Parkinson-White syndrome.

Prevention of tachycardia initiated from the right atrium. With tachycardia initiated from the high right atrium, preventive train stimulation at the same site 50 ms after the tachycardia-initiating stimulus did not prevent tachycardia induction in any patient, because the atrial effective refractory period in these patients was >140 ms. When train delivery was shifted toward diastole, tachycardia was prevented in all patients as soon as the train extended beyond the atrial effective refractory period of the tachycardia-initiating stimulus (Fig. 2A to C).

The site of collision of the tachycardia-initiating and preventive wave fronts could be different, depending on the timing of the preventive train. With "early" prevention (Fig. 2B) the tachycardia-initiating wave front had not yet activated larger parts of the ventricles when the preventive wave front (A_p) was blocked in the specific conduction system and traveled anterogradely through the accessory pathway (pre-excitation in the corresponding QRS complex) to collide with the tachycardia wave front on the ventricular level below the AV node. In contrast, "late" prevention (Fig. 2C) allowed the tachycardia wave front to almost complete its orthodromic circus movement through the specific conduction system and ventricles (no pre-excitation in the corresponding QRS complex). However, close to the insertion site of the accessory pathway, fusion occurred with the preventive wave front. When prevention failed because of resetting of the tachycardia, the change of the atrial activation sequence indicated wave front collision on the atrial level.

Prevention of tachycardia initiated from the coronary sinus. With tachycardia initiated from the coronary sinus, preventive train stimulation attempted from the high right atrium was successful in only 4 of the 10 patients. These four were patients whose accessory pathway was located on the right side of the heart. In the other six patients, who had a left-sided accessory pathway, progressively delayed train stimulation of the high right atrium, with the train extending beyond the effective refractory period of the right atrial response to the tachycardia initiating stimulus, never prevented tachycardia (Fig. 3A and B). However, when the train was delivered to the coronary sinus, after the tachycardia initiating stimulus with sufficient delay to achieve single left atrial capture, tachycardia induction was prevented (Fig. 3C).

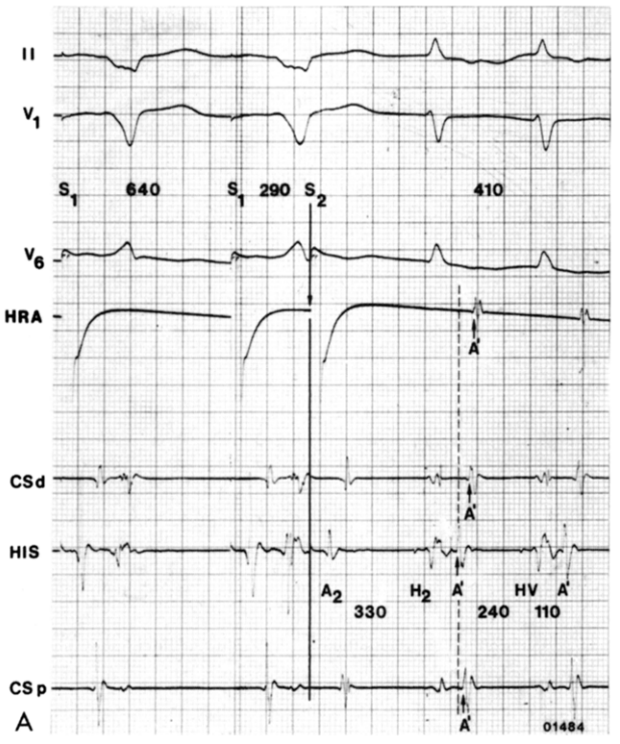
From the data given in Table 1 for these six patients one can see that in each patient the sum of right atrial effective refractory period and conduction times from left to right atrium and vice versa exceeds the initial tachycardia interval at the coronary sinus (for example, Patient 7: [190 + 70 + 60] ms >270 ms). This accounts for the failure of right atrial preventive stimulation when the tachycardia is induced from the coronary sinus.

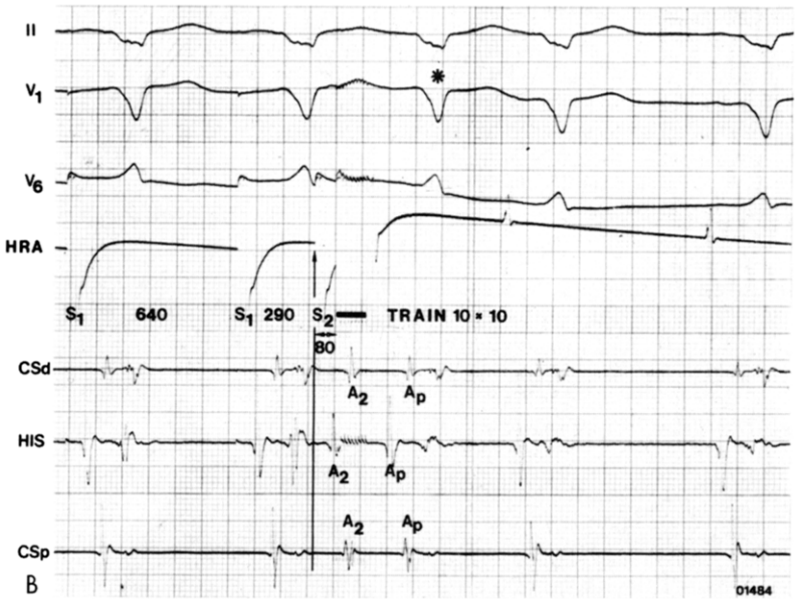
Discussion

Train stimulation. Ultrarapid train stimulation has been shown to terminate an ongoing tachycardia more effectively than does single extrastimulation (4). On the basis of a previous study (2) confined to the right atrium that had clarified the basic mechanism of AV tachycardia prevention with single extrastimuli, we have used the train approach for the prevention of tachycardia induction in patients with an AV accessory pathway. To more closely simulate the real life situation, tachycardia was initiated from both the

right and the left atrium. Train prevention of tachycardia induction was attempted at both atrial sites.

Mechanism of tachycardia prevention. Preventive stimulation at the site of tachycardia induction was successful in all patients, regardless of the site of the accessory pathway. Because the preventive stimulus can immediately follow the effective refractory period of the tachycardia-initiating stimulus and impulse conduction within the reentrant circuit necessarily exceeds atrial refractoriness, collision of the tachycardia-initiating wave front with the preventive wave front must occur within the reentrant circuit. The site of wave front collision depends on the conduction velocities and refractory periods within the reentrant circuit as well as the timing and site of preventive train stimulation. In our study, with successful prevention, wave front collision occurred at various sites on the ventricular level. Train stimulation with wave front collision on the atrial level always reset the tachycardia.





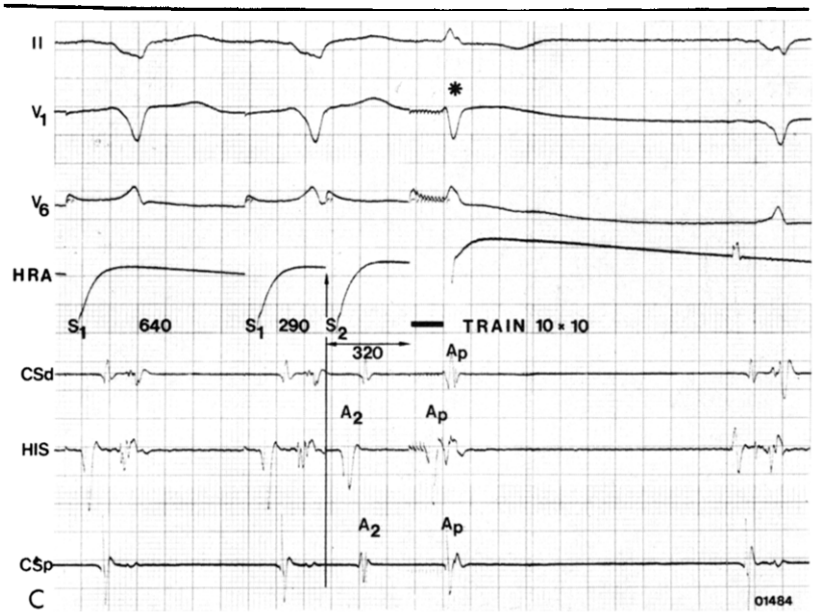


Figure 2. Patient 3. **A,** Induction of reentrant tachycardia from the high right atrium with a single extrastimulus (S_2) at a coupling interval of 290 ms during basic drive pacing (S_1S_1 = 640 ms). The **broken vertical line** denotes earliest retrograde activation (A′ at bundle of His). **Small arrows** indicate retrograde atrial activation (A′) sequence: bundle of His (HIS) - proximal coronary sinus (CSp) - distal coronary sinus (CSd) - high right atrium (HRA), indicative of a right-sided anteroseptal accessory pathway. Tachycardia cycle length is 410 ms. Shown are three surface electrocardiograms (II, V_1 and V_6) and four intracardiac leads. **B,** Tachycardia induction sequence as in **A.** However, induction of tachycardia is prevented by single atrial capture (A_P) after delivery, at the high right atrium of a train of 10 extrastimuli 80 ms after S_2. Atrial capture is achieved by the last train stimulus, which is the only one to extend beyond the effective refractory period of 160 ms. The **asterisk** denotes a pre-excited QRS complex. See text for details. **C,** Tachycardia induction is still prevented by delivery of a train at the high right atrium 320 ms after S_2. The **asterisk** denotes a normal QRS complex without pre-excitation. The activation sequence of the atrial capture (A_p) is identical to that of A_2, indicating anterograde activation.

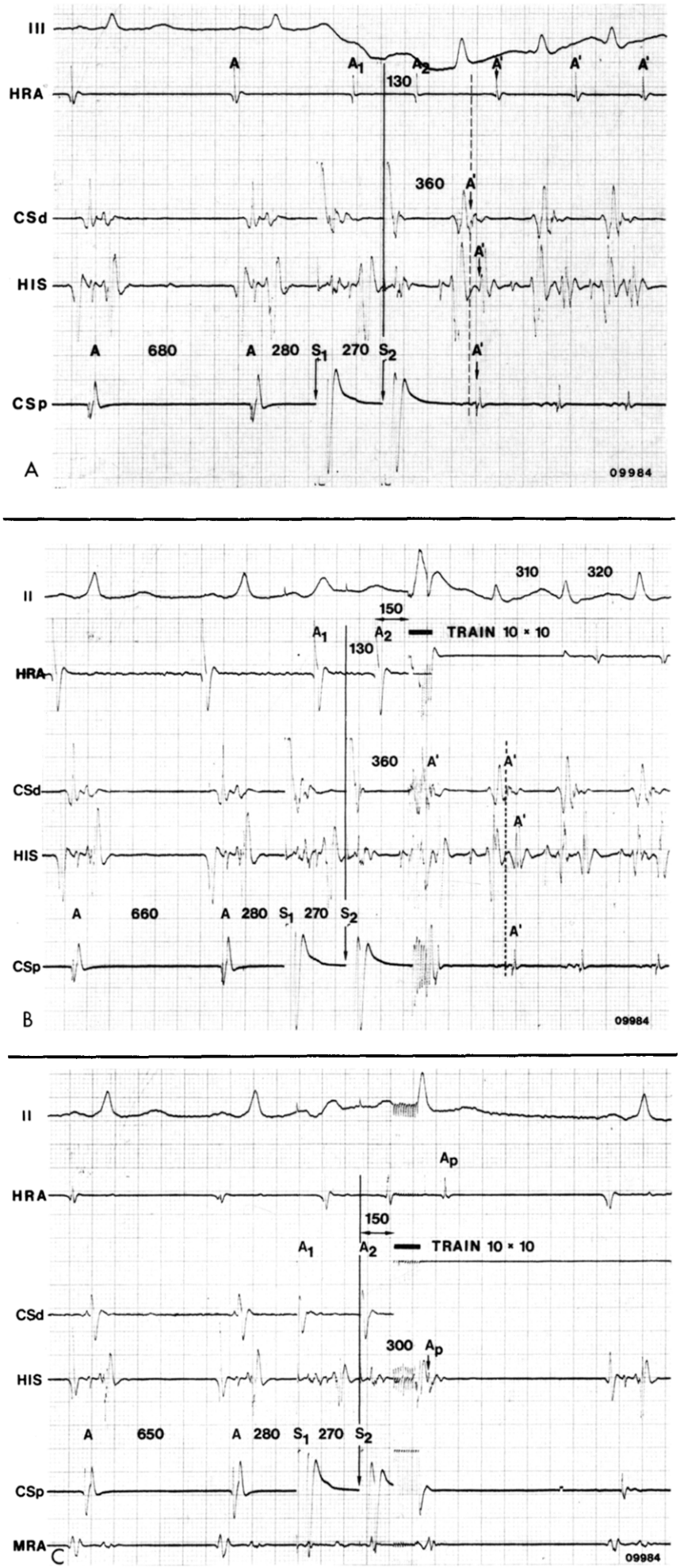


Figure 3. Patient 9. **A,** Initiation of reentrant tachycardia from the proximal coronary sinus (CSp) with two extrastimuli (S_1, S_2) at 280 and 270 ms, respectively, during sinus rhythm (sinus cycle length 680 ms). The **broken vertical line** denotes the earliest retrograde activation (A′ at the distal coronary sinus 360 ms after S_2). The **small arrows** indicate a retrograde atrial activation (A′) sequence (CSd - CSp - HIS - HRA), indicative of a left lateral accessory pathway. Note that S_2 takes 130 ms to reach the high right atrium. Shown are surface ECG lead III and four intracardiac leads. **B,** Tachycardia initiation sequence as in **A.** Train stimulation at the high right atrium (effective refractory period 200 ms) 150 ms after the right atrial response (A_2) to the tachycardia-initiating stimulus (S_2) fails to prevent tachycardia induction. The train stimulus to achieve right atrial capture, 330 (= 130 + 200) ms after S_2, would have needed another 130 ms (adding to a total of 460 ms after S_2) to travel to the distal coronary sinus (CSd). This exceeds the initial tachycardia interval of 360 ms by 100 ms. **C,** Tachycardia initiation sequence as in **A.** Train stimulation at the distal coronary sinus 150 ms after A_2 (= S_2) prevents tachycardia induction by single left atrial capture (A_P), observed in the His bundle electrogram 300 ms after S_2. Abbreviations as in Figure 2.

Single capture in train stimulation. Even though 10 extrastimuli are delivered to the atrium, preventive train stimulation as performed in this study is a single-capture technique. The stimuli are timed so closely (they cover an interval of 90 ms) that only one of them (the first to extend beyond the atrial effective period of the preceding tachycardia-initiating stimulus) achieves atrial capture (Fig. 1D) and thus renders the atrium refractory to the remainder of the train stimuli. This stimulus then enters the reentrant circuit to interfere with and prevent tachycardia induction. Because the right and left atrial effective refractory periods in our patients ranged from 160 to 250 ms and 210 to 330 ms, respectively, preventive train stimulation 150 to 250 ms after, and at the site of, tachycardia induction was always successful.

Prevention failure due to conduction delay. Failure of preventive train stimulation was encountered when the preventive wave front was delayed for too long to invade the reentrant circuit in time. Such was the case in six patients with a left-sided accessory pathway in whom the tachycardia was initiated from the coronary sinus and prevention attempted from the high right atrium. The initiating wave front, while entering the anterograde limb of the reentrant circuit (the specific conduction system in orthodromic tachycardia), is delayed by the interatrial conduction time before it depolarizes high right atrial myocardium for the duration of the effective refractory period. Thus, any right atrial stimulus would be effective in preventing an orthodromic tachycardia only if it collided with the tachycardia-initiating wave front in the retrograde limb of the reentrant circuit. In our six patients, the overall conduction delay of the preventive stimulus—comprising the conduction time from the left to the high right atrium plus the right atrial effective refractory period plus the conduction time from the high right atrium back to the left atrium—turned out to be too long for the stimulus to enter the reentrant circuit. Conduction delay is reduced by moving the site of preventive stimulation closer to the anatomic site of the reentrant circuit; thus prevention from the coronary sinus was successful in these patients.

Train characteristics and atrial fibrillation. The duration and configuration of the train of stimuli were arbitrarily chosen. Trains of 6 stimuli 20 ms apart will in principle be as effective as the trains of 10 stimuli 10 ms apart used in our study. The train may also be lengthened, yet the possibility of multiple atrial capture by train stimuli must be strictly avoided, so as to not reset the tachycardia or increase the risk of inducing atrial fibrillation. Within our stimulation protocol atrial fibrillation was not induced in any patient.

Preventive train stimulation for other types of tachycardia. The principle of preventive train stimulation should also work in the setting of other types of reentrant supraventricular tachycardia, such as AV node tachycardia (5), because the inherent mechanism of tachycardia prevention in any case is premature invasion of the reentrant circuit. A recent observation by Miller et al. (6) suggests that reciprocating ventricular tachycardias may be prevented by programmed ventricular stimulation.

Interaction of extrastimuli for induction of reentrant tachycardia. The mode of action in preventive stimulation clearly demonstrates that the interaction of multiple extrastimuli is crucial for the induction of a tachycardia mediated by anatomically well defined pathways. It is generally believed that an increase in the number of extrastimuli increases the chance of inducing tachycardia, as has been demonstrated (7) in patients with and without documented ventricular tachycardia. This may be true in patients without the electrophysiologic substrate for reentrant tachycardia. However, as can be inferred from our study as well as from the observation of Miller et al. (6), in patients with such a substrate (either AV or ventricular) it is likely that multiple stimuli may extinguish each other, depending on their relative timing. This has not yet been studied systematically in patients with documented tachycardia in whom stimulation is generally terminated when the tachycardia is induced, with no further increase of the number of extrastimuli.

Antitachycardia pacemaker considerations. Train stimulation, as opposed to single beat extrastimulation, appears to be a promising mode to be incorporated into an implantable antitachycardia pacemaker system, because timing of the preventive extrastimulus is no longer as critical and, also, the length of the train may be adjusted to compensate for spontaneous changes of sinus cycle length and refractoriness. However, this latter problem needs further investigation. Moreover, preventive train stimulation should be carried out close to or within the reentrant circuit.

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